Saint Bartholomew's Hospital Journal

VOL. LXV DECEMBER 1961 No. 12

ST. BARTHOLOMEW'S HOSPITAL JOURNAL

Editor : A. M. POLLOCK

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DECEMBER

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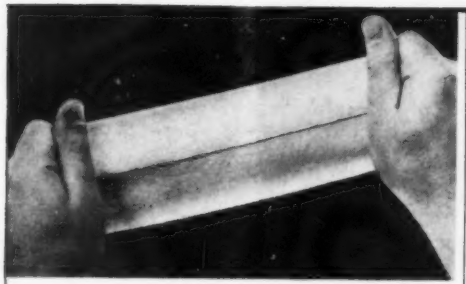

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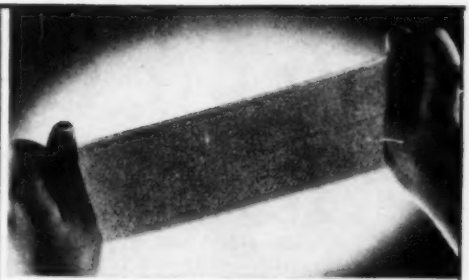

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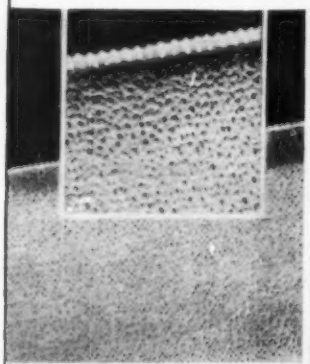

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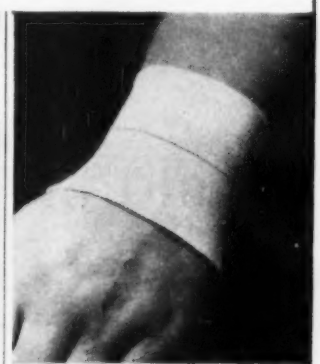

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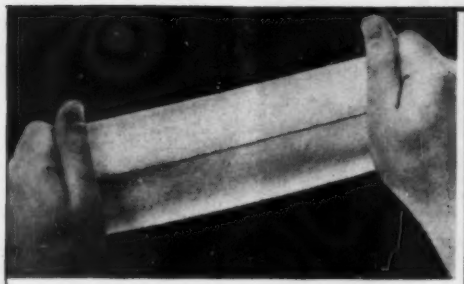

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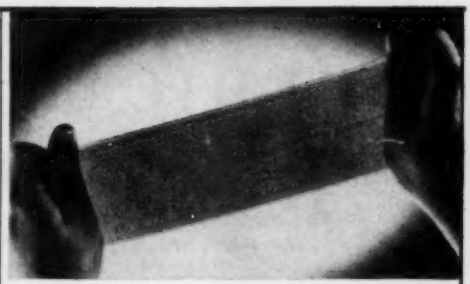

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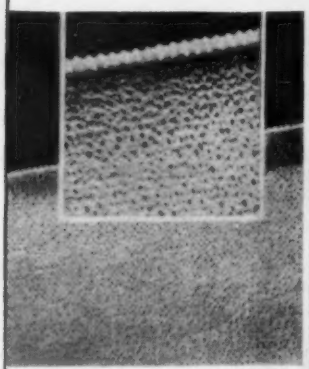

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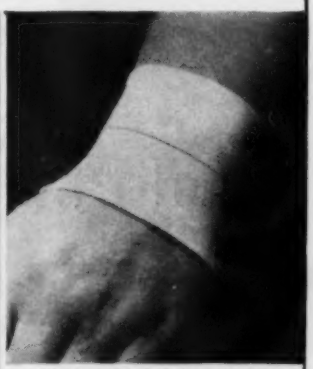

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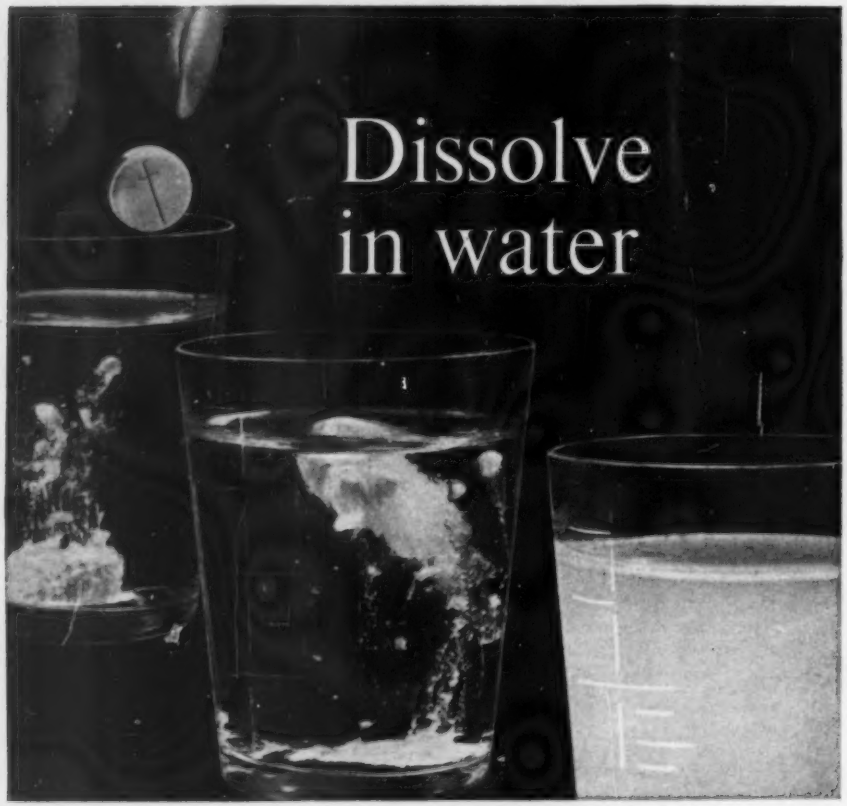

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ST. BARTHOLOMEW'S HOSPITAL JOURNAL

Vol. LXV, No. 12 DECEMBER 1961

Editorial

A RECENT DEBATE in the House of Lords on the Medical Services aired many of the old clichés and tired platitudes known only too well to members of the profession. The miscalculations of the Willink Commission and the present need for a ten per cent increase in student doctors are now old history. Apart from a ritual ventilation of the problems their Lordships (and their Ladyships) had little concrete to add. One of the questions that remained unasked was where are we going to find additional doctors? Are we going to find them? This is a matter to which, for a moment, we might well turn our thoughts.

The medical profession has always been, loosely speaking at any rate, a conservative one. To the newer kind of man, demanding high rewards for little work, this could prove off-putting. So, perhaps, we ought to make ourselves more attractive — professionally speaking, that is. In a world whose values are rapidly being undermined by the slogans of Madison Avenue, where virtues like duty, vocation and service are made to sound like an old music-hall joke, perhaps we too ought to concern ourselves with the status symbol and the right brand image. Handled properly the stethescope should be a status symbol par excellence. It is all a matter of handling.

A letter received recently by the Dean makes one wonder if things have not gone rather wrong somewhere; and if the general impression of a medical college is not rather distorted. Written by a sixth form public schoolboy seeking admission it asks the following: —

"1. Games Facilities. I am interested in playing golf. Would it be difficult to get to a reasonable club? How far is it, and what does it cost? 2. Music facilities. Are there any facilities available for learning the piano?"

The Dean might have replied that clock-golf whiled away the weary hours in Out-Patients, that the Abernethian Room was large enough to swing a mashie in, and that the senior surgeons of the hospital frequently offered free extra-mural instruction in approach, grip, cut and slice. "Music While You Operate" is a great favourite in the theatre where a large concert grand is always at students' disposal. Violin stringing a speciality. If the Dean did not reply in this vein (and we have no reason to presume that he did) might not another possible ten per center have given up thoughts of a medical career in favour of the green and the green-room?

Advertising has always been frowned upon by Medicine. But when it comes to recruitment just how high-principled can one afford

Editorial—*Continued*

to be? The Army has surely shown the way —"JOIN THE ARMY AND SEE THE WORLD." Why not—"BE A DOCTOR AND GET THE INSIDE STORY"? Or something with more subtle implications like "DOCTORS READ THE TIMES", or something flattering, "WHICH TWIN IS THE SURGEON", embarrassing, "SOMEONE ISN'T A DOCTOR", or even a direct poster in the Kitchener tradition, "WHOSE FINGER ON THE PULSE?" With the psychologists on our side everything is possible; there are no emotions we could not exploit—the basic ones best of all. The time might even come when the G.P. could again be as much a friend of the family as the "Man from the Pru". At present he is in jeopardy of becoming a name on the list in the post office, or a service telephone number.

Means would have to be devised for coping with the vast influx of recruits such advertising campaigns would encourage. Correspondence courses are the obvious answer. "Learn to be a DOCTOR in six easy years" . . . "study by your own fireside." "Interesting cases brought to your *very* door by Hospitals on Wheels Inc.—Our motto 'Keep the Patient Moving'." " . . . Deep-freeze specimens obtainable from your nearest delicatessen," etc. The possibilities of such a scheme are infinite.

But as readers will no doubt have observed there is one snag to all this. Our aim must be to encourage people to become doctors and not, unnecessarily, to become patients. An overawareness of sickness and its needs could have disastrous results. The man in the street, let it be remembered, is only a doctor at heart in so far as he is a potential patient.

Flippancy apart, the reasons for adopting a medical career are hard to define, and hard to publicise. Often they are personal; always they sound trite when given verbal expression. Once the Government has made up its mind how to accommodate more students, it is then up to the students to find themselves. Parkinson's Law will no doubt do the rest.

Engagements

BANWELL—BOWER.—The engagement is announced between Gerald Stuart Banwell, F.R.C.S. (Edin.), and Miss Gillian Bethune Bower.

FOX—NORTON.—The engagement is announced between Dr. Geoffrey Charles Fox and Miss Jill Pauline Norton, S.R.N.

GARROD—ONSLOW-FREE.—The engagement is announced between Dr. James Anthony Garrod and Miss Sally Anne Onslow-Free.

GLYN—CLIVE.—The engagement is announced between Dr. Alan Glyn, M.P., and Lady Rosula Caroline Windsor Clive.

WHITTARD—DAUNCEY.—The engagement is announced between Dr. Brian Ralph Whittard and Dr. Shirley Frances Dauncey.

Marriages

BRISTOW—SWIFT.—On 29th July, Ronald Frank Bristow to Julia Ann Swift.

RACK—LAGEARD.—On 8th July, Henry D. Rack to Violette M. E. Lageard.

Births

CHARLTON.—On 21st October, to Jennifer (née Price), wife of Dr. Clive Charlton, a son (Simon Rupert).

GODRICH.—On 28th October, to Chloe (née Mathieson), and Dr. John Godrich, a son (Jeremy).

SINGER.—On 9th November, to Mary (née Hilder), and Dr. Geoffrey Singer, a son (John Geoffrey), brother for Alison, David and Claire.

Deaths

GRIFFITH.—On 29th October, Adrian Nicholas Griffith, aged 33. Qualified 1952.

KING.—On 21st October, Lt.-Col. Harold Holmes King, C.I.E., M.B., B.S. Qualified 1908.

Appointments

University of Manchester

Dr. P. J. Collard, who held the chair of bacteriology in the University College of Ibadan, Nigeria, has been appointed professor of bacteriology and director of the department of bacteriology, and will take up his duties in Manchester in April, 1962.

Change of Address

Surg.-Lt. D. A. LAMMIMAN, R.N., H.M.S. Raleigh, Torpoint, Cornwall.

Calendar

DECEMBER

Wed.	27 to Fri. 29	"Pot Pourri" at the Cripplegate Theatre.
Sat.	30—On duty:	Dr. G. W. Hayward Mr. A. W. Badenoch Mr. R. W. Ballentine

JANUARY

Sat.	6—On duty:	Dr. A. W. Spence Mr. E. G. Tuckwell Mr. T. B. Boulton
Sat.	13—On duty:	Medical and Surgical Units Mr. G. H. Ellis
Sat.	20—On duty:	Dr. R. Bodley Scott Mr. A. H. Hunt Mr. F. T. Evans
Sat.	27—On duty:	Dr. E. R. Cullinan Mr. C. Naunton Morgan Mr. R. A. Bowen

MISS KATHLEEN TURNOCK

Miss Kathleen Turnock, on 31st October, left us to retire to her newly-acquired home in the country—the countryside she loves so much—after many years of devoted professional service to the Hospital.

Miss Turnock was in the unique position of having been awarded the prize of books at the end of her first year, and also the Gold Medal at the completion of her training in 1931.

Then followed her appointments as Theatre Pink, Night Sister, Sister of Rahere and Sister of Lawrence. She had to leave for a short period, but returned to us in 1939 as second Assistant Matron. Shortly after her return, she went to Hill End, where she spent many happy years. In 1949 she was appointed Deputy Matron, the post she has held until now.

Administration these days in a large teaching hospital such as ours is an arduous task for all concerned; there is so much unseen work, and, like a snowball, it grows and grows. Miss Turnock, as Deputy Matron, was outstanding in the sympathy and understanding she extended to all. She was ever ready to lay down whatever she was doing and listen and give advice to all who asked for it.

In an age characterized by its lack of standards, perhaps her most valuable contribution has stemmed from her high sense of personal duty, based on those ultimate values which do not change.

We know that Miss Turnock's life in "retirement" will be a busy one, as there is always much to attend to in a house and garden, and village activities will no doubt claim their full share of her time. However, we hope that she will not become so involved with her new life that her visits here will need to be curtailed.

F.O.

Fifty years ago

"It is a platitude to insist that a good teacher *nascitur non fit;* it is equally a platitude to maintain that an eminent clinician with the highest academic distinctions may not be able to teach at all. But, quite apart from these considerations, is not 'examination-medicine' quite different from 'medicine', and may not the more recently qualified man be better able to teach students, because he is more *au courant* with examination wrinkles, and is also nearer the intelligence of his pupils? Of course, in this connection we do not mean **any** recently qualified man; we mean the exceptional man, who has been qualified a few years only, and from whom the savour of examinations has not yet departed."

"Examiners are drawn from those who are farthest removed from examinations, so that the seniors should be the best teachers to defeat examiners, and examination wrinkles should be unnecessary and useless."

"One may regard the ideal teacher as a man who has advanced sufficiently along the path of knowledge to enable him to plant himself at the meeting point of many cross-roads. Having become familiar with all these paths he is able to direct a timid traveller along any one of them. He has not yet emulated the older explorer who has followed one of the paths to its termination there to remain; and who whilst knowing the most minute details of the district he has reached, has long since forgotten the steps of the journey he has traversed and the very existence of the cross-roads and the parts to which they lead."

The Abernethian Society

On Thursday, 26th October, 1961, the Society met for a symposium on Degenerative Arterial Disease.

In the chair was Dr. G. W. Hayward, who opened the meeting and introduced the speakers in turn, Dr. D. Weitzman, Prof. G. W. Taylor, and Dr. R. Finlayson.

Dr. Weitzman discussed the role of calculating lipids in the aetiology of atheroma and showed that although there was a high pre-β lipid concentration in atheroma, it was also high in the blood of patients with other diseases. He closed by saying that although oestrogens had been used in the U.S.A. to prevent atheroma, he did not recommend this treatment here.

Prof. G. W. Taylor discussed peripheral arterial disease and described a new technique used to determine the viability of skin, particularly important in cases of gangrene, to decide whether amputation is necessary and where it

should take place. The method involved the patient breathing in 100 per cent. O_2, then by subcutaneous electrodes, if the current was raised 100 per cent., the skin was considered viable.

Dr. Finlayson showed slides of atheroma in 900 post-mortem specimens at London Zoo. His subjects were varied and slides excellent. The common factor in this atheroma in birds and humans was age, but he found no evidence of thrombosis and little evidence of coronary atheroma or myocardial infarcts in the animals.

The meeting was then open for discussion and questions and Dr. Hayward closed by discussing the aetiology of atheroma. Mr. John Goldman gave a vote of thanks to the panel and chairman and the meeting was declared closed.

Notice board observers will have seen the appearance of a new style of poster introduced by the newly-formed Staff Christian Fellowship, it is in respect of their inaugural Open Meeting on 4th December, to be addressed by Mr. Stuart Mawson, and with Mr. John Beattie in the chair. The subject, "The Voluntary Adoption of Faith," seems singularly appropriate to the occasion.

We welcome with great pleasure this Fellowship of the senior and lay staffs of the hospital and sincerely hope it may be the means of fulfilling some of the spiritual requirements of those on the House, in the labs and behind the scenes. We wish it every blessing and believe that it will serve to deepen still further the basic ethical practice of Bart's along the line of its Christian heritage. B.J.S.

Christian Union

A new feature has appeared in the life of the Christian Union over the past eighteen months. During this time, missionary societies have been asked for the names of Bart's doctors working with them; the response of these societies, together with a few of our own personal contacts with overseas posts, has enabled us to formulate a type of "Who and Where is Who". Those in touch with our opposite number in "The London" will be familiar already with this very useful type of compilation.

The idea is to utilise this for establishing contacts between members and doctors overseas in order to have up-to-date news about the various aspects of their work and the problems involved. This is to enable the several small prayer groups that have been formed to pray informatively for the missionaries in their areas.

All the information, however, will be circulated amongst members and contributors. In this way it is also hoped to link up the overseas folk with the Alma Mater and with each other in prayer.

Should any ex-Christian Union member like to have this report, and subsequent annual supplements when published, or if they have the names of unattached missionaries whom we are unlikely to have contacted, would they write, please, to the Missionary Secretary.

Last Month

At the beginning of last month students this side of the meat market were still reminiscing about the quality of the Halloween Ball held in College Hall on the last Friday of the previous month. The occasion was, I am told, as smooth as its organiser. In the light of this success Student Union sponsored Ball Committees would do well to find flair to lighten their stodgier qualities.

The Rugger tour took place during the second week of last month and the Bart's team (which included several preclinicals) won a game and lost two. Often one still hears comments that rugger at Bart's is not what it used to be. This is, of course, true and sad, but since authoritative opinion has abolished the "Rugby Scholarships" there is little that can be done except to sit back and pray for fifteen brains with brawn. Big Brother might have thought twice before demonstrating the stiff competition of the modern medical course by refusing to carry our sporting passengers any longer.

The Nursery plays were in the third week of November. They were smart productions and certainly an evening's entertainment. However, they were perhaps an unfortunate trio in that they were all of a peculiar, odd, modern type—A. P. Herbert, Simpson and Ionesco all thrown together, left one decidedly confused.

S.C.-S.

CANCER OF THE LARYNX

By Alan Fuller

This paper will discuss the clinical behaviour of cancer of the larynx and the methods available for its treatment, using for illustration sixty patients first seen in this hospital between 1948 and 1952.

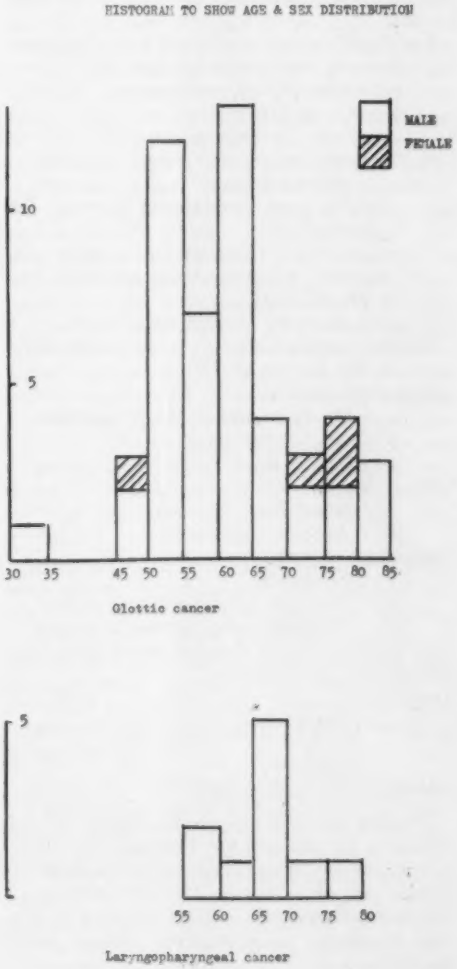
The prognosis of the clinical behaviour of a cancer, the type of treatment selected and the tumour's probable response to treatment are dependent on the site of the tumour, its degree of malignancy, local extension and the absence or presence of metastases.

Equally the diagnosis of a cancer will depend on its symptoms which are referable to its site, its size, local spread and possible metastases. It is in this respect that cancer of the larynx may be divided into those that produce early symptoms and those that are silent in their earlier stages.

Tumours of the vocal cord produce their only symptom, hoarseness, early, as they are on the margin of the glottis. Hoarseness lasting more than two weeks, particularly in middle age, demands inspection of the larynx. The growth is characteristically found in the middle third of the cord, frequently extending to the anterior commissure and rarely to the posterior third. It may appear as a fusiform swelling, a granuloma or a warty growth. Keratinisation may be so marked that the mass looks white (the "snow carcinoma"). Local deep extension may be revealed by impaired mobility of that cord. The differential diagnosis at this stage is tuberculosis and syphilis. A chest X-ray and a blood examination for syphilis must be carried out. Coronal tomography of the larynx may reveal a subglottic mass which cannot always be seen on indirect laryngoscopy. The next step is to perform a direct laryngoscopy usually under general anaesthesia although local anaesthesia can be used. This will afford a thorough examination of the larynx and removal of part of the tumour for histological examination.

A carcinoma originating from below the vocal cord will have to extend to a far greater degree to produce interference with the voice. Subglottic growths carry a graver prognosis than cordal growths because they present later, and because lymphatic spread occurs early via the abundant subglottic lymphatics to the paratracheal lymph nodes.

A carcinoma of the supraglottic portion of the larynx arises in a symptomatically silent part of the body. Symptoms are not produced at the site of the tumour until it becomes ulcerated and infected, so producing pain and dysphagia or until its mass is sufficient to interfere with the airway. The presenting symptom in the three supraglottic carcinomas reported here was dyspnoea in two and hoarseness in one.



The laryngopharyngeal margin is also a silent part of the body and symptoms are produced only when ulceration occurs. Lymphatic metastases are often a presenting symptom. The presenting symptoms in ten cases were soreness of the throat in seven patients and one patient each with huskiness, dysphagia and a cervical swelling.

Although no two patients with malignant disease can be said to be identical it is extremely useful in practice to place them in categories dependant on the tumour site, local spread and the presence or absence of metastases. The International Union against Cancer (Paris, 1958) agreed to the following International Classifications of Cancer of the Larynx.

Laryngeal cancer is classified by Regions:

(a) Larynx, which is subdivided into
subglottic (inferior region).
glottic (middle region),
subglottic (inferior region).

(b) Laryngopharyngeal margin, which is subdivided into
anterior part (suprahyoid part of the epiglottis),
middle part (junction of 3 folds, viz. epiglottis, pharyngo-epiglottic fold, aryepiglottic fold),
posterior part (aryepiglottic fold).

Further subdivisions are made in the divisions of the larynx as follows:—

Supraglottic region:
(a) Posterior aspect of the epiglottis,
(b) Ventricular band,
(c) Ventricle.

Glottic region:
(a) Vocal fold,
(b) Anterior commissure.

Subglottic region:
(a) Subglottis,
(b) Anterior subcommissural region,
(c) Posterior subcommissural region.

The patients seen in this five year period were distributed as follows:—

Larynx		50
Supraglottic	3	
Glottic	38	
Subglottic	9	
Laryngopharyngeal margin		10

Further information on the extent of the disease is provided in the International Classification by ascribing categories to the local extension of the tumour, the involvement of regional lymph nodes and the presence of distant metastases when present. This is coded as follows:—

Tumour=T
T1=tumour limited to one region and not affecting mobility.
T2=tumour invading one region with limitation of mobility, or tumour invading two regions.
T3=tumour invading more than two regions, or with destruction of the thyroid or cricoid cartilages.
T4=tumour involving a neighbouring structure (organ).
Adenopathy=N
N0=no palpable enlarged lymph nodes.
N1=homolateral, mobile palpable nodes.
N2=bilateral, mobile palpable nodes.
N3=homolateral or bilateral fixed nodes.
Metastases=M
M=metastases at a distance.

Using the above categories of T, N and M the tumour may be placed in the following stages:—

Stage I=T1, N0.
Stage II=T1, N1 *or* T2, N0.
Stage III=T1, N2 *or* T2, N1 *or* T3, N0 *or* T3, N.1, *or* T3, N.2.
Stage IV=T1 or 2 or 3, N.3 *or* T1 or 2 or 3, M *or* T4.

At diagnosis the patients were distributed as follows:—

Glottic	
Stage I	29
Stage II	8
Stage III	1
Subglottic	
Stage II	5
Stage III	4
Supraglottic	
Stage I	1
Stage II	1
Stage III	1
Laryngopharyngeal margin	
Stage I	2
Stage II	0
Stage III	3
Stage IV	5

Carcinoma of the larynx is most commonly primary, secondary carcinomas are rare. Histologically the majority of cases are squamous celled; adenocarcinomas and basal cell carcinomas are rarities. The degree of differentiation may vary between highly keratinised tumours with cell nest formation and anaplastic growths. In this series histological confirmation was obtained as follows:—

Glottic cancer	35 out of 38
Subglottic cancer	9 out of 9
Supraglottic cancer	3 out of 3
Laryngopharyngeal margin cancer	6 out of 10

In all cases the carcinoma was squamous celled. The relationship between the cellular structure of the tumour as shown on biopsy and survival of the patient is displayed in the following tables.

Glottic cancer

Histology	Survival 1 yr.	1-3 yrs.	3-5 yrs.	5 yrs. +
No biopsy	1			2
Carcinoma in situ		1	1	2
Well differentiated		1	2	7
Moderately well differentiated	1		2	4
Poorly differentiated	1	1		6
Not graded	1	1	1	3

Subglottic cancer

Histology	Survival 1 yr.	1-3 yrs.	3-5 yrs.	5 yrs. +
Well differentiated	1			
Moderately well differentiated		1		2
Poorly differentiated	1			2
Not graded	1			1

Supraglottic cancer

Histology	Survival 1 yr.	1-3 yrs.	3-5 yrs.	5 yrs. +
Well differentiated				1
Poorly differentiated	1			
Not graded				1

Laryngopharyngeal margin cancer

Histology	Survival 1 yr.	1-3 yrs.	3-5 yrs.	5 yrs. +
No biopsy	3			1
Poorly differentiated		3		1
Not graded	2			

The grading of the tumour from the biopsy does not give an accurate prognosis of the behaviour of the tumour, though highly cellular growths often do well.

The methods of treatment available are radiotherapy or surgery or a combination of both. Radiation treatment can be given by external radiation or interstitial radiation. X-rays are applied externally and are generated from a high voltage source. The depth of penetration through the skin depends on the voltage used at their source. During the period under review this hospital was particularly fortunate to have a deep X-ray machine capable of generating X-rays from a million volt source. The greater the voltage used in generation the greater the depth dose for a given skin dose, and it is the skin dose that can be tolerated without producing local tissue necrosis or constitutional change that is a major factor limiting treatment. The conventional voltage at this time was 250,000 volts so that a million volt machine was a great advance. It has been found that the patient can accept more radiation if the total dose is fractionated over a period of 30-40 days. In general it is planned to give a tumour dose of 6,000 roentgen in six weeks, but the clinical reaction may involve variation of this target.

Interstitial treatment by radium needles was pioneered in this country by Dr. Finzi and Mr. Harmer of this hospital. In this, under general anaesthesia, a window is cut in the thyroid cartilage and also in the upper part of the cricoid cartilage. Into this window radium needles are inserted vertically in a palisade over the internal layer of perichondrium beneath which lies the vocal cord. About four to six needles containing 2 or 1 mgm. of radium are used. The intensity of radiation emitted by radium falls off by the square of the distance. The vocal cord is nearer the perichondrium of the thyroid cartilage anteriorly than it is posteriorly. Thus it can be seen that the needles must be placed closer together at the posterior part of the window if a uniform dose is to be achieved. After the needles have been placed in position the probable dose rate is calculated by the physicist and the needles are removed when the tumour has received about 8,000 roentgen. This usually takes between 6-7 days. As the needles are implanted deep to the skin the limitation imposed by skin sensitivity does not arise and cartilage also vulnerable to radiation has been removed.

After successful treatment by radiation the voice returns to normal and the vocal cord returns to an almost normal appearance. In fact, if this does not occur within two months of the end of the treatment, it is a sure sign that all is not well. The only change produced in the vocal cord being the development after some months of occasional telangiectasia along its surface.

The surgical procedure used in the treament of vocal cord cancer is laryngofissure and local excision of the vocal cord. Afterwards the defect in the larynx is filled by granulation tissue, which becomes covered by epithelium and converted to fibrous tissue. A fibrous tissue band develops in approximately the position of the excised vocal cord so that phonation can be obtained by compensatory movement of the

remaining cord across the mid-line. The voice, however, is never normal and may be very poor.

Laryngectomy can be considered the final court of appeal in laryngeal cancer. It involves the removal of the whole larynx, the upper limit must extend from the vallecula, in front of the epiglottis, around the aryepiglottic fold to the back of the arytenoids. The hyoid bone or at least its body must be removed because forward extension to the pre-epiglottic space is frequent. The lower limit of excision is the trachea at least 0.5 cm. below the growth. The pharyngeal defect is closed and the trachea brought forward into the neck as a permanent tracheostomy. The disability after laryngectomy is not incompatible with a normal working life. In this hospital it is usual to ask the patient's employers to write to him before operation offering to keep his job or to find a suitable substitute. Oesophageal speech is acquired by most patients. In this a basic voice-tone is produced by half-swallowing air into the hypopharynx and upper oesophagus and releasing it by a controlled eructation. This tone is acted upon by the various speech moulds (tongue, palate, lips, etc.), to produce speech. Various ingenious artificial larinqes have been developed. All of them have to be controlled manually thus limiting the physical agility of the patient. Patients may become depressed after laryngectomy and one patient in this series died as a late result of a suicide attempt. Eighteen months after laryngectomy for a recurrence of cordal cancer, he was found in a park with a bullet wound in his head. This did not kill him and he lived for another four months with a hemiplegia before succumbing to acute broncho-pneumonia.

The results of treating sixty patients are shown in the following tables:—

Glottic cancer: *Stage I*
Treated by 1,000 Kv. D.X.R. alone.

Cases	*Survival* 3 years	5 years
14	12 (86%)	9 (64%)

Deaths to date 8

Cause of death	*Survival*
Carcinoma larynx	9 mths.
Carcinoma larynx	4 yrs. 2 mths.
Carcinoma larynx	4 yrs. 5 mths.
Carcinoma bronchus	9 yrs. No recurrence
Carcinoma pancreas	5 yrs. 7 mths. No recurrence
Myocardial degeneration	3 yrs. 9 mths. No recurrence
Congestive heart failure	2 yrs. 8 mths. No recurrence
Cerebral arteriosclerosis	10 yrs. 8 mths. No recurrence

Glottic cancer: *Stage I*
Treated by 1,000 Kv. D.X.R. initially recurrence treated by LARYNGECTOMY.

Cases	*Survival* 3 years	5 years
3	3 (100%)	2 (66%)

Deaths to date 1

Cause of death	*Survival*
Suicide	4 yrs. 4 mths. No recurrence

Glottic cancer: *Stage I*
Treated by laryngofissure excision.

Cases	*Survival* 3 years	5 years
4	4 (100%)	1 (25%)

Deaths to date 4

Cause of death	*Survival*
Congestive heart failure	3 yrs. 2 mths. No recurrence
Carcinoma bladder	3 yrs. 9 mths. No recurrence
Carcinoma bronchus	3 yrs. 6 mths. No recurrence
Haemorrhage from chronic gastric ulcer	10 years No recurrence

Glottic cancer: *Stage I*
Treated by Finzi-Harmer radium implant.

Cases	*Survival* 3 years	5 years
7	7 (100%)	6 (85%)

Deaths to date 3

Cause of death	*Survival*
Carcinoma bronchus	4 yrs. 1 mth. No recurrence
Carcinoma prostate	6 yrs. 2 mths. No recurrence
Carcinoma hepatic ducts	6 yrs. No recurrence

One patient developed a carcinoma of the opposite vocal cord five years after treatment, this was treated by radium implant. He survived another five years without recurrence.
The patient who died from primary hepatic cancer developed a local recurrence at eight months. This was successfully treated by laryngofissure and local excision.

Glottic cancer: *Stage I*
Treated by sequence of D.X.R., laryngofissure and radium implant.

Case	*Survival*
1	7 years+

D.X.R. followed by recurrence at 10 months.
Laryngofissure excision followed by recurrence after 22 months.
Radium implant with survival for 5 years+ with disease present.

Glottic cancer: *Stage II*
Treated by 1,000 Kv. D.X.R. alone.

Cases	*Survival* 3 years	5 years
5	2 (40%)	1 (20%)

Deaths to date 4

Cause of death	*Survival*
Carcinoma larynx	1 yr. 1 mth.
Carcinoma larynx	4 mths.
Carcinoma bronchus	1 yr. 3 mths. No recurrence

Perforation D.U. 4 yrs. 2 mths. No recurrence

Glottic cancer: *Stage II*
Treated by 1,000 Kv. D.X.R. with treatable recurrences.

Cases	*Survival* 3 years	5 years
2	2 (100%)	1 (50%)

(a) Recurrence at 11 mths. treated by laryngofissure excision — Survival afterwards 6 yrs. 11 mths.
(b) Recurrence at 14 mths. treated by laryngectomy — 2 yrs. 3 mths.

Deaths to date
2

Cause of death	*Survival*
Carcinoma larynx	3 yrs. 5 mths.
Cerebral thrombosis	8 yrs. 10 mths.

No recurrence

Glottic cancer: *Stage II*
Treated by Finzi-Harmer radium implant.
Case 1 *Survival* 9 years+. No recurrence.

Glottic cancer: *Stage III*
Treated by laryngectomy.
Case 1 *Survival* 4 months.
Cause of death: Asphyxia due to aspiration of a plug of mucus. No recurrence.

Subglottic cancer: *Stage II*
Treated by 1,000 Kv. alone.
Case 1 *Survival* 7 months.
Cause of death: Carcinoma larynx.

Subglottic cancer: *Stage II*
Treated by laryngectomy.

Cases	*Survival* 3 years	5 years
2	2 (100%)	2 (100%)

1 recurrence at 6 years 6 months treated by 250 Kv. D.X.R. to neck: survival afterwards 9 months.
Deaths to date
1

Cause of death	*Survival*
Carcinoma larynx	7 yrs. 3 mths.

Subglottic cancer: *Stage II*
Treated by Finzi-Harmer radium implant.

Cases	*Survival* 3 years	5 years
2	2 (100%)	2 (100%)

1 recurrence at 6 months, treated by block dissection of cervical glands followed by 250 Kv. D.X.R. to the neck: survival afterwards 7 years+.

Subglottic cancer: *Stage III*
Treated by 1,000 Kv. D.X.R. alone.

Cases	*Survival*
2	4 months and 5 months.

Cause of death
Carcinoma of the larynx.

Subglottic cancer: *Stage III*
Treated by laryngectomy.

Cases	*Survival* 3 years	5 years
2	1 (50%)	1 (50%)

1 recurrence at 2 months, treated by 250 Kv. D.X.R. to neck: survival afterwards 11 months.
Cause of death: carcinoma of larynx.

Supraglottic cancer: *Stage I*
Treated by 1,000 Kv. D.X.R.
Case 1 *Survival* 8 years 6 months.
Cause of death
Myocardial degeneration. No recurrence.

Supraglottic cancer: *Stage II*
Treated by laryngectomy.
Case 1 *Survival* 12 years+. No recurrence.

Supraglottic cancer: *Stage III*
Case 1 *Survival* 4 days after tracheostomy.
Cause of death
Carcinoma of larynx.

Larvngopharyngeal margin cancer: *Stage I*
Treated by 1,000 Kv. D.X.R.

Cases	*Survival* 3 years	5 years
2	1 (50%)	1 (50%)

Death to date
1
Cause of death Carcinoma epiglottis *Survival* 1 yr.
The surviving case had in addition diathermy excision of the epiglottis two months after completing the course of radiation.

Laryngopharyngeal margin cancer: *Stage III*
Treated by 1,000 Kv. D.X.R.

Cases	*Survival* 3 years	5 years
3	1 (33%)	1 (33%)

3 *recurrences*
(a) at 6 months treated by pharyngo-laryngectomy (elsewhere). Survival afterwards: 6 months.
(b) at 7 months treated by diathermy amputation of epiglottis. Survival afterwards: 9 months.
(c) at 6 years 6 months given palliative treatment only. Survival afterwards: 2 months.
Deaths
3
Cause of death
Carcinoma larynx in all three.

Laryngopharyngeal margin cancer: *Stage IV*
1 case died before receiving treatment.

Treated by D.X.R.

Cases	*Survival* 3 years
4	nil

Deaths
4

Cause of death	*Survival*
Carcinoma larynx	6 mths.
Carcinoma larynx	9 mths. (2 cases)
Carcinoma larynx	11 mths.

Survival by Stages of Cancer of the Larynx.

Glottic cancer

	Cases	3 *years*	5 *years*
Stage I	29	27 (93%)	19 (65%)
Stage II	8	5 (62%)	3 (38%)
Stage III	1	0	
All stages	38	32 (84%)	22 (58%)

Subglottic cancer

	Cases	3 *years*	5 *years*
Stage II	5	4 (80%)	4 (80%)
Stage III	4	1 (25%)	1 (25%)
All stages	9	5 (55%)	5 (55%)

Supraglottic cancer

	Cases	3 *years*	5 *years*
Stage I	1	1 (100%)	1 (100%)
Stage II	1	1 (100%)	1 (100%)
Stage III	1	0	
All stages	3	2 (66%)	2 (66%)

Laryngopharyngeal margin cancer	*Cases*	*3 years*	*5 years*
Stage I	2	1 (50%)	1 (50%)
Stage III	3	1 (33%)	1 (33%)
Stage IV	5	0	
All stages	10	2 (20%)	2 (20%)

Cause of death in 37 patients		
Carcinoma larynx		20
Second primary cancer		8
(Bronchus	4	
Pancreas	1	
Bladder	1	
Prostate	1	
Hepatics ducts	1)	
Heart disease		4
Cerebral thrombosis		2
Haematemesis		1
Perforation duodenal ulcer		1
Asphyxia		1
Suicide		1

It is interesting to note the large proportion of patients who have died from their original disease. So far it comprises more than half those who have already died, but even if none of those surviving die from carcinoma of the larynx it will still be the cause of death in one third. The development of a second carcinoma in the respiratory tract is another feature of note.

I should like to acknowledge the help of Mr. Capps in preparing this paper and would like also to thank the Surgeons to the Throat Department for allowing me access to their notes.

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LOCKED TWINS

By Humphry Ward

FEW OBSTETRIC TEXTBOOKS fail to discuss this rare and dangerous complication of multiple pregnancy. Its infrequency is such that many obstetricians never see a case in a lifetime. Van Braun in two Vienna Clinics gives the incidence as 1 in 90,000 deliveries or approximately 1 in 1,000 twin births.

The following case was recently seen at Rochford General Hospital: —

Mrs. E. C., a primigravida, aged 23 years, whose estimated date of delivery was 6th September, 1961, was first seen on 10th August, 1961. She was then thirty-six weeks pregnant, but the size of the uterus was that of full term.

11*th August.* On admission to hospital, her general condition was fair. The mucuous membranes were pale. The B.P. was 145/85, with marked oedema of the legs but with no albuminuria. The girth at the umbilicus was 41½ in. There was no hydramnios. An X-ray examination confirmed the diagnosis of a twin pregnancy—the leading foetus was a breech, left sacro-anterior, and the second was presenting by the vertex. The pelvis was normal. The haemoglobin was 65 per cent. A course of "ferrivenin" 100 mgm. daily was given for five days.

Up to this time the pregnancy had been normal. The patient had been given oral iron, but this had failed to prevent an iron-deficiency anaemia—a common complication of multiple pregnancy. She was allowed home after ten days.

31*st August.* On re-admission, the B.P. was 130/90, with moderate oedema of the legs. The foetal positions were unchanged.

4*th September.* A medical induction (castor oil, soap enema and bath) yielded no result.

9*th September.* A repeat was also unsuccessful.

13*th September.* She was given an intravenous Dextrose 5 per cent containing 1 unit "Syntocinon" increasing to 3 units (per pint of Dextrose 5 per cent). This was continued for the rest of the day with poor results.

15*th September.* A vaginal examination was performed under sterile conditions.

The breech was presenting but was high and above the pelvic brim. The cervix admitted one finger only. The conditions were unsuitable for a surgical induction, so stretching the cervix and sweeping the membranes had to suffice at this stage.

16*th September,* 8 *p.m.* The first stage of labour commenced, with moderate contractions every five minutes.

17*th September,* 12 *mid-night.* The cervix was two fingers dilated.

7 *a.m.* The temperature was 99°F. and rose to 100°F. by

10 *a.m.* There was acetone in the urine. An intra-venous drip was given with one pint Dextrose 10 per cent and thereafter Dextrose 5 per cent.

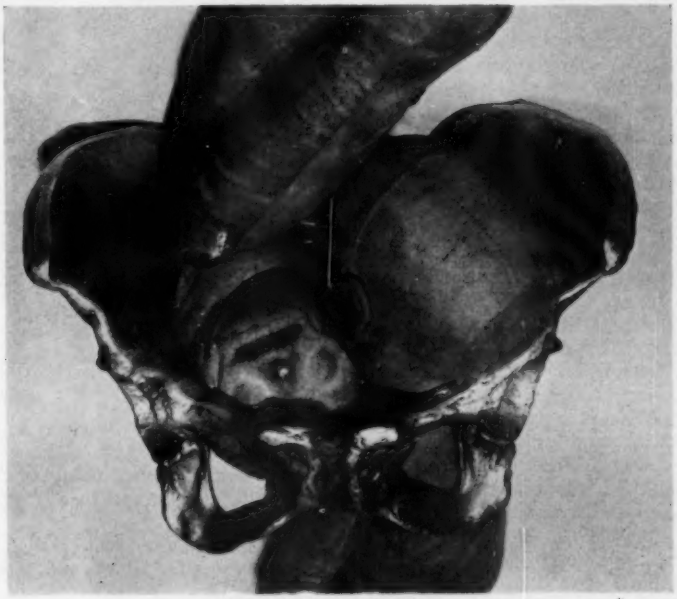
Progress was fair with strong regular contractions every three minutes, but the relaxa-

tions were not complete, suggesting a "high resting tone" of the uterine muscle.

6 *p.m.* "Pethilorfan" 150 mgm. and "Sparine" 50 mgm. was given intramuscularly.

8 *p.m.* Despite the earlier sedation, the patient was very distressed and not resting. Gas and air was given to help control the urge to push. Vaginally a rim of cervix was felt all round.

10.30 *p.m.* "Pethilorfan" 100 mgm. was given intramuscularly but had little effect on a very restless patient. Both foetal hearts could still be heard. On examination there was extreme tenderness over the lower segment.



Breech-Vertex locking.

Vaginally, the breech was visible and the cervix was fully dilated. The position was left sacro-anterior.

11.30 *p.m.* One leg was delivered under a pudendal nerve block. Episiotomy was done.

11.45 *p.m.* The trunk followed. Attempts to bring down the head failed and under general anaesthesia a diagnosis of locked twins was made. Neither foetal heart could now be heard.

18*th September,* 12.15 *a.m.* The consultant arrived.

On examination — the cervix was fully dilated. The head of the second twin lay on the right side of the pelvic cavity in the left occipito-posterior position. The head of the first twin lay above this and was deflexed with the occiput prominently palpable above the symphysis pubis. The heart beat was absent. The posterior parietal boss of the second twin was below the sacral promontory, but the anterior boss was thrust in the neck of the first foetus.

Delivery of Twin 1.

The second amniotic sac was ruptured artificially. An attempt to disimpact the second head upwards failed.

Two facts contributed to this failure: —

(1) Delivery of the first twin had proceeded too far to permit any upward movement. The anterior shoulder had escaped from below the symphysis pubis before locking became apparent.

(2) The contraction and retraction of the uterus.

Decapitation of the first twin and forequarter amputation with embryotomy scissors was done. The body, minus the right, i.e. posterior arm, which remained connected to the head and neck, was then delivered. It was then possible to displace the head and remaining quarter of the first foetus above the head of the second.

Delivery of Twin 2.

A direct application of Neville-Barnes for-

ceps was attempted but was unsuccessful.

Kielland's forceps were applied to the head of the second twin and the occiput rotated to the directly posterior position. Delivery was completed easily with the Neville-Barnes forceps. The condition of this twin suggested asphyxial death before attempted delivery. The placenta was removed manually.

The twins were female, uniovular and weighed 5 lb. 5 oz. and 5 lb. 7 oz. respectively.

The patient was pyrexial for some days in the puerperium but clinical and bacteriological investigation failed to show any cause for this. She was discharged for home nursing on the fifth day, her condition being satisfactory.

Comment.

Locked twins are rarely predicted before the onset of labour and is usually not discovered until the time of delivery. *Aetiology :* Nicholson[5] suggests that deficiency of liquor amnii, uniovular twins, deflexion of the head of the leading twin and a large pelvis with small foetuses may all be contributory factors. He points out that there is no single element, but rather a number of conditions which predispose to locking.

Williamson[7] agrees that there is usually more than one contributory factor.

Holland and Bourne[2] comment on the predominance of primigravidae in Lawrence's[3] series. There were twenty-three primigravida out of twenty-eight cases. It should be noted that in the present case the patient was a primigravida, the twins were uniovular and that oligohydamnios was a striking feature. The time of rupture of the first amniotic sac was not known because of the absence of any gush of liquor at any time. When the second sac was ruptured artificially there was only a slight trickle of mecomium stained liquor. The connection between this condition of oligohydramnios and prolongation of pregnancy to 10 days beyond the expected date of delivery is a point of interest.

In the Lawrence series, the most common type of locking was Breech and Vertex (15). This variety is much more serious than that in which the two forecoming heads become impacted (9), as this is seldom of any consequence as the second twin can usually be pushed up out of the way. In a third type, locking between vertex and a transverse presentation, three cases were recorded. Finally the rarest of all; impacted breeches, when a variable number of limbs appear at the vulva (1).

The foetal mortality was 39 per cent; for the leading twin it was 57 per cent. There were no maternal deaths. Of the fifteen Breech-Vertex impactions, thirteen were still-born. A successful case was reported by Greig[1], who was able to disimpact the two heads without a general anaesthetic. Both children lived. This may have been a case which in the United States of America is known as " Collision " rather than " locking ". (Swann[6].) In the same article a case of locking in triplets is recorded, in which impaction occurred between the first triplet, a breech and the second, a vertex. All the triplets survived.

Treatment depends on the condition of the leading foetus and the type of locking. The principle is that of disimpaction and assisted delivery of the leading twin where possible. When this is not possible, decapitation of the first foetus is necessary, so that the second may be delivered alive. It appears that traction of the breech and the bringing down of the arms should not be attempted before every effort has been made to disimpact the heads.

A method of delivery is described by Kimball and Rand[4]. The second twin is delivered first using Piper forceps and the breech subsequently by manual flexion. The patient (gravida 3 parous 2) is described as short, light of structure with a normal pelvis. The twins weighed 4 lb and 4 lb. 12 oz. respectively. In her second pregnancy she had a 7 lb. 10 oz. girl.

Williamson advocates Caesarean section if the patient's condition allows. He describes the management in his Breech-Vertex impaction. Caesarean Section has no advantages unless the second twin is known to be alive. Holland and Bourne point out that section is a far safer manoeuvre than a traumatic vaginal delivery.

A case of locked twins is described with some of the causative factors and possible treatment.

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A CASE OF GALACTOSAEMIA

Being the entry "Proxime Accessit" to the Bentley Prize, 1961

By Sylvia Watkins

Galactosaemia is a rare, inborn error of metabolism. Probably the first published account of the disease was that given by von Reusse in 1908: in a discussion on urinary sugars, he pointed out that galactosuria was rare in comparison with glycosuria and lactosuria. He described a case of "Alimentary galactosuria due to toxic disturbances of the liver". In 1917, Göppert described "Galactosurie mit Milchzuckergabe bei angeborenem familiärem chronischem Leberleiden", and he gave a detailed description of a patient with hepatomegaly, jaundice and galactosuria, who responded well to a lactose free diet.

Both these early descriptions are characteristic of the disease. Typically, the galactosaemic infant is normal at birth, but symptoms appear soon after milk feeds are started. Food refusal, listlessness, vomiting, diarrhoea, loss of weight and jaundice (which at first appears to be a prolongation of the neonatal physiological jaundice), are characteristic presenting symptoms. The spleen and liver are enlarged, and the urine is found to contain galactose, and later, amino acids, casts and protein; the amount of galactose present is directly proportional to the lactose intake, and is therefore greater in breast-fed babies than in artificially fed infants (since there is more lactose in breast milk than in cows' milk or dried preparations). If the untreated child survives long enough, he develops convulsions, mental deficiency and nuclear cataracts. Gradually hepatic failure develops, with ascites, oedema, and haemorrhages into the skin and mucous membranes. The most frequent causes of death in the untreated patient are hepatic failure and intercurrent infections. Fortunately, however, the course of the disease may be arrested by giving the patient a lactose free diet. Later in life, the patient often develops a gradually increasing tolerance to small amounts of lactose, though return to a normal diet is never possible.

Case Report: C.J.P.: male infant.

C.J.P. was the second child born to normal parents, following a normal pregnancy and delivery his weight at birth on 24th May, 1960, was 7 lb. 10 oz. On the second day of life he started to vomit, and became jaundiced the next day, his liver being then one finger's breadth palpable. The following day, the jaundice was deeper, and the liver further enlarged; his urine was found to reduce Benedict's reagent, but not Clinistix. A presumptive diagnosis of galactosaemia was made, and later confirmed by chromatographic studies of his urine. He was put on a lactose free diet, consisting of Wanderlac and sugar, giving 360 calories per day. At one week of age, the galactose had disappeared from his urine, the jaundice had faded, the liver receded, but he was still vomiting, and was not gaining weight. He was admitted to Lucas when three weeks old, weighing 6 lb. 4 ozs.; on examination at this time, he looked small and thin, and had a palpable liver and spleen, but seemed otherwise normal. His urine contained both galactose and amino acids. He developed a Proteus urinary infection, which responded well to treatment with Tetracyclines.

At this stage, severe feeding difficulties were encountered on the Wanderlac diet, and his progress was poor. "Glue feeds," consisting of Robinson's groats, arachis oil, eggs, vitamins and minerals, were tried, but this gave him such severe gastro-intestinal disturbances that oral feeding had to be stopped for two days, during which he was given intravenous Hartmann's solution and 5 per cent. dextrose.

On 8th July, at 6½ weeks of age, his weight was still only 5 lb. 10¼ oz. Wanderlac was started again: he gained ½ lb. in two weeks, but then diarrhoea and vomiting started again. It was pointed out that Wanderlac (which is supposed to be lactose free) actually contained traces of lactose. As an alternative, Nutramigen was tried; this is an enzymatic hydrolysate of casein, with added glucose, fats, vitamins and minerals. He thrived on this extremely well, gaining 3 lb. in four weeks.

He was discharged on 30th August, 1960, aged 14 weeks, on a diet of Nutramigen supplemented by Abidec and Rose Hip syrup. At 5½ months he was taking, in addition to the Nutramigen, groats, beef broth, liver, peas, beans, and carrots; his weight was then 16 lb. 6 oz. When last seen on 2nd January, 1961, he seemed well and happy; there was no evidence of cataract, nor of any of his earlier symptoms.

This, then, is an example of a case of

galactosaemia which was recognised early in life. After an initial stormy period, during which he reacted violently to even minute traces of galactose, he settled down well on a strictly lactose-free diet. He now shows none of the characteristic signs or symptoms of the disease, and there is every hope that he will develop normally in every way, except, of course, that his dietary restrictions must remain.

There have been three other diagnosed cases of galactosaemia in St. Bartholomew's Hospital since 1947. Two of them (S.D. and P.A.), both girls, born in 1957 and 1959 respectively, seem to be well at present, keeping to a strictly regulated diet, based on Wanderlac. S.D., now aged four, is small for her age, and her speech is rather poor, but there is no real evidence of mental retardation, nor of cataract, either in her or in P.M.A., who is now two. The third patient, J.L., also a girl, born in 1956, is now being followed up at another hospital, but when last seen, she was doing well on diet based on Galactomin.

The symptoms and signs of the four children are summarised in the table below.

DISCUSSION

The main clinical features of galactosaemia may be divided into the following groups for the purpose of discussion: renal, hepatic, gastro-intestinal, and other miscellaneous disturbances.

Disorders of Renal Function

Galactosuria is a finding common to all cases of the disease. Amino-aciduria is usually also present (and was noted in C.J.P., S.D. and J.L.). However, whereas galactosuria appears only in association with a raised serum

TABLE SHOWING THE SIGNS AND SYMPTOMS IN THE FOUR PATIENTS.

	C.J.P. (Male)	S.D. (Female)	P.M.A. (Female)	J.L. (Female)
BIRTH WEIGHT	7 lb. 10 oz.	6 lb. 4 oz.	7 lb. 10 oz.	8 lb. 1 oz.
AGE OF PRESENTATION	2nd day	9th day	5th day	8th day
PRESENTING SYMPTOMS	Vomiting Jaundice	Loss of weight Vomiting Jaundice	Food refusal Loss of weight Jaundice	Vomiting Jaundice
SYMPTOMS				
Lethargy	√	√	√	√
Jaundice	√	√	√	√
Vomiting	√	√	√	√
Food refusal	√	√	√	√
Total weight loss	2 lb.	1 lb.	1 lb. 7 oz.	
Time (in weeks) till B.W. regained	10½ weeks	3½ weeks	5 weeks	
SIGNS Enlarged liver	1 f.b.	2 in. below R.C.M.	Enlarged	Enlarged to umbilicus
Enlarged spleen	√	√	√	
INVESTIGATIONS DURING FIRST ADMISSION				
Bilirubin	15.5 mg. %	5.9 mg. %		7.9 mg. %
Galactosuria	√	5 Gm. %	√	√
Amino-aciduria	√	√		√
Thymol turbidity		4.5 units		
Alk. phosphatase		48 K-A units	21 K-A units	
Pseudocholinesterase		37 units		
Haemoglobin	68%	46%		76%
GALACTOSE TOLERANCE INDEX IN PARENTS				
Father		299		163
Mother		107		127
FOOD BEST TOLERATED	Nutramigen	Wanderlac	Wanderlac	Galactomin

galactose, there is no increase of serum amino acids to account for the amino-aciduria. Hence there are two possible mechanisms either of which could explain this phenomenon:

(a) a congenital abnormality of tubular function, associated with lowered renal threshold for amino acids.

(b) a temporary inability to re-absorb amino acids, due to the action of some toxic agent on the tubular cells.

If a galactosaemic patient is given galactose for one day only, amino-aciduria does not occur; but if given for longer periods, amino-aciduria appears within 5-6 days, and disappears within a few days of returning to a galactose free diet. These findings exclude the possibility of a congenital defect, in which case the amino-aciduria would be present all the time, regardless of the galactose content of the diet. "We must conclude that the amino-aciduria . . . is a later change induced by the primary abnormality" (Cushworth, Dent and Flynn). The findings in fact suggest that the phenomenon is due to a reversible tubular dysfunction, probably resulting from a toxic metabolic product, several days being required for recovery from the toxic effect. The nature of the supposed toxic agent will be discussed later.

Other renal abnormalities include metabolic acidosis and proteinuria (which disappear on a galactose free diet). However, sodium, potassium and water excretion are not altered, which suggests that either only one part of the tubule is affected, or that the general disturbance is insufficient to affect these basic functions. (Komrower, Schwarz, Holzel and Golberg.)

Hepatic Function

Liver function is severely impaired in untreated cases, the earliest symptom being the persistent jaundice in the neo-natal period. All four children in this series were jaundiced, and raised serum bilirubin levels were recorded in C.J.P., S.D. and J.L. Liver function tests performed in S.D. and P.M.A. gave abnormal results, shown in the Table. All four patients recovered from their jaundice when a lactose free diet was started. This is in accordance with the general rule that galactosaemic jaundice is reversible provided that the diet is instituted within the first few weeks of life. We may conclude that this early hepatic dysfunction is of purely functional origin (i.e. there is no organic pathology in the liver); in this respect it is analogous to the renal disturbances, and may similarly be attributed to a reversible toxic action on the liver cells, resulting from the abnormal metabolism. However, in fatal cases, post mortem examination often reveals varying degrees of fatty change, cellular degeneration, or even diffuse hepatic fibrosis, indicating that in the later stages of the untreated disease, the hepatic dysfunction is due, in part at least, to structural alterations in the liver, and as such, is irreversible.

Gastro Intestinal Disturbances

Three of the Bart's patients (C.J.P., S.D. and J.L.) presented with vomiting, and all four children refused their food, vomited and lost weight during the early stages. These symptoms cannot be attributed to any pathological lesion. However, Komrower, writing about one of these patients (P.M.A.) stated his belief that "children often refuse substances that are harmful to them, and that 'a matter of taste' may be a diagnostic indication". Just what makes these patients vomit is not clear; but Komrower's extremely sensible view is probably sufficient explanation of why they vomit and refuse their food.

Other Lesions

Untreated cases of galactosaemia are mentally deficient, suffer from convulsions, and sooner or later develop nuclear cataracts. None of the Bart's patients have shown any sign of these complications. The convulsions are thought to be due to a combination of a toxic metabolic effect (similar to that mentioned in connection with hepatic and renal dysfunction) and the severe hypoglycaemia which occurs in these patients, if galactose is present in their food. This hypoglycaemia is probably due to preferential absorption of galactose, and reciprocal inhibition of glucose absorption. (James and Leak.) The incidence of the fits may be reduced by giving large quantities of dextrose. The hypoglycaemia in these patients does not appear to be important in the aetiology of the hepatic and renal dysfunction, since other causes of hypoglycaemia are not associated with disturbances of this type.

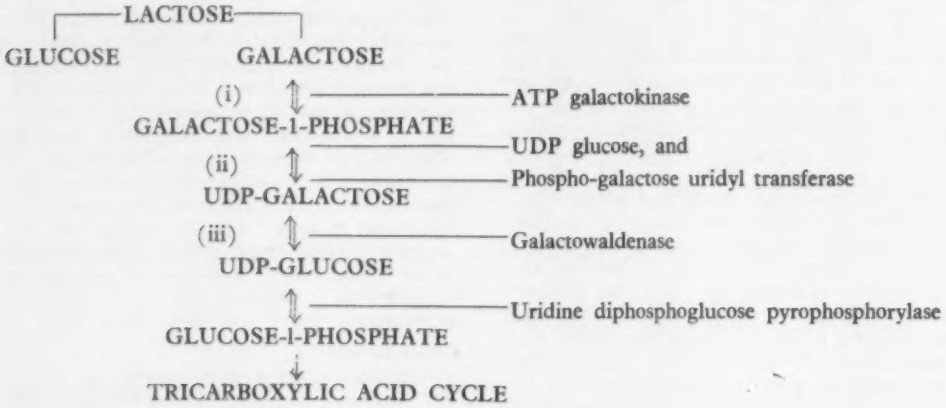
There is evidence that in the presence of high experimental plasma galactose levels in rats, galactose metabolites accumulate in the lenses. These are probably responsible for disturbing lenticular metabolism, later resulting in cataracts. This is yet another aspect of the toxic metabolic effect.

This discussion gives rise to a number of questions which must be considered in relation to the clinical, biochemical and therapeutic problems of galactosaemia. What is the nature of the metabolic block ? What substance exerts

a toxic effect on the cellular metabolism of various tissues ? How do the patients gradually develop increased tolerance to galactose ? How is the disease acquired ? What are the best lines of treatment available ? These problems will now be discussed.

The Enzymic Block

The metabolic path of galactose is as follows:—



Galactosaemic patients accumulate galactose and galactose-1-phosphate in their body fluids and cells. This suggests that the metabolic block must lie at either reaction (ii) or (iii), i.e. there must be a deficieny of either P-gal-uridyl transferase, or galactowaldenase; or alternatively there might be an anti-enzyme, or lack of some essential co-factor. Kalckar, Anderson and Isselbacher, working with erythrocytes, showed that P-gal-uridyl transferase (which is present in the cells of normal subjects) is lacking from those of galactosaemic patients. The enzyme galactowaldenase is present in both the normal and the abnormal cells. These workers could find no evidence of an anti-enzyme, nor of a missing co-factor, and concluded hat the lack of P-gal-uridyl transferase was the lesion responsible for the condition. Deficiency of this enzyme was demonstrated in one patient of this series (P.M.A.).

The "Toxic" Agent

It has been suggested above that many of the clinical features are due to disturbances of cellular function by a "toxic metabolic product". Schwarz, Golberg, Komrower and Holzel, working on erythrocytes, showed that the galactosaemic cell metabolism (as demonstrated by oxygen uptake) is inhibited in the presence of galactose. This inhibition cannot be due directly to the galactose (which actually increases the metabolic rate of normal cells); therefore it must be due to a metabolite, probably galactose-1-phosphate, which does accumulate owing to the enzyme deficiency. These workers suggest that the metabolic inhibition might be due to competition between glucose-1-phosphate and galactose-1-phosphate for the essential co-enzyme glucose-1:6-diphosphate, thus:

Galactose-1-phosphate + glucose-1:6-diphosphate→Galactose-1:6-diphosphate + glucose-6-phosphate thereby gradually transforming the normal co-enzyme into galactose-1:6-diphosphate, which is useless in glucose metabolism. It seems likely, then, that the effect of accumulating galactose-1-phosphate is an inhibition of normal glucose metabolism, which is responsible for producing much of the energy required by the cell. This inhibition can easily account for the failure of so many cells to function normally.

The Development of Galactose Tolerance

As patients increase in age, their ability to metabolise galactose increases. This was noticed already by Göppert in 1917, when he remarked that "Je jünger das Kind ist, desto grösser dürfte die Schwierigkeit sein". This phenomenon suggests that there is an alternative metabolic pathway for galactose; some galactose metabolism certainly takes place in older patients, as shown by galactose excretion studies. Isselbacher, using $1-C^{14}$ labelled galactose, recently proved the existence of such

a path, probably the following:

Galactose-1-phosphate + UTP ⟺ UDP-galactose + PP

He showed that the level of the necessary enzyme (UDP-gal-pyrophosphorylase) increases with age in normal subjects, and he suggested that the symptoms of galactosaemia were maximal in infancy, because then there is "both a pathological absence of P-gal transferase, and a physiologically feeble uridine-diphospho-galactose-pyrophosphorylase". As the latter enzyme's activity increases with age, the patients' galactose metabolism improves, in spite of the continued absence of the former.

The four Bart's patients are as yet too young to show any significantly increased tolerance to galactose, although they are all managing to take a rapidly increasing variety of foods. For example, P.M.A. is now able to drink Wanderlac in a concentration which she could not take initially, probably because of the traces of lactose present.

Inheritance of the Disease

The familial incidence of the condition suggests that it is inherited. Galactose tolerance tests have been carried out on the parents and other relatives of galactosaemic children: several had abnormal tolerance, though without any clinical manifestations of the disease. This suggests that clinical galactosaemia is inherited as a homozygous recessive characteristic, whilst heterozygosity confers abnormal galactose tolerance, without any clinical signs (Holzel and Komrower). Galactose tolerance was tested in the parents of S.D. and J.L., with the following results:

	Galactose index
Mr. D.	299
Mrs. D.	107
Mr. L.	163
Mrs. L.	127
Normal range	0-160

These results are not of great significance, but they do exhibit some degree of abnormal galactose metabolism in the parents of these patients.

More recently it has been shown that estimation of the level of the enzyme P-gal-uridyl transferase is a more sensitive index for demonstrating the abnormality in both homo- and heterozygotes. Huang, Hugh-Jones and Hsia studied both enzyme levels and galactose tolerance tests in heterozygous carriers, and concluded that "the frequent occurrence of this condition in siblings, and among the offspring of consanguineous matings, together with its equal distribution in both sexes, suggest that galactosaemia is probably transmitted by a single autosomal recessive gene".

Treatment

Göppert, in 1917, recommended replacing the "Milckzucker" by other sugars, such as sucrose or glucose. Today the management of these patients is based on a lactose free diet in the form of powder milk substitutes. Patients vary greatly in their tolerance of any given make of powder: "Galactomin", "Wanderlac", and "Nutramigen" were found, by a process of trial and error, to suit J.L., P.M.A. and S.D., and C.J.P. respectively. In fact, they all contain traces of lactose, and Komrower has pointed out that, as in the case of P.M.A., full strength Wanderlac may be badly tolerated, perhaps because "at full strength the amount of galactose children receive is just sufficient to build up a pathological level of galactose-1-phosphate in the cells". This very probably applies to other makes likewise, and could well account for the large number of distressing upsets in early life.

Later on, the diet must be based on rice flour and certain tinned foods, whilst substances such as Casilan, Robinson's groats, Farex and many others must be avoided. The gastro-intestinal upset suffered by C.J.P. on his "glue feeds" was probably due to the groats, which contain stachyose, of which galactose is a component. Another important aspect of this problem is that many common therapeutic agents, such as Vitamin C and Penicillin V tablets, are made up in lactose, and therefore cannot be used.

Recently (1960), Pesch, Segal and Topper have shown that steroids stimulate galactose catabolism both in vivo and in vitro; also, progesterone materially reduces the incidence of cataracts in galactose fed rats. In vitro experiments have shown that the effect is due to stimulation of the enzyme of the alternative pathway. Prepubertal galactosaemic patients given progesterone before an injection of C^{14} labelled galactose, had a greater output of $C^{14}O_2$ (i.e. greater galactose catabolism) than those not receiving progesterone. The authors of this work do not claim that progesterone can replace the present treatment, but they do suggest that it might be useful during exacerbations of the disease, when the development of cataract and mental retardation seem to be progressive.

Prognosis

The prognosis for these patients depends mainly on the age at which the lactose free diet is started, and on whether or not it is strictly adhered to. In those patients whose diet is started early, and carefully managed, the signs

and symptoms disappear rapidly, and most children can be expected to develop normally: C.J.P., S.D. and P.M.A. seem at present to be well and normal, and the prognosis for them is good. However, almost all children in whom treatment is delayed even as little as six weeks, are mentally retarded, and later develop cataracts.

Remaining Problems

The biochemical nature of the disease has been elucidated; symptomatic treatment has been studied and successfully applied. But the fact remains that the life of the galactosaemic patient becomes progressively more difficult as he grows older: in our civilisation, all forms of social contact are centred round the sharing of food and drink, much of which, in this part of the world, contains milk. The resulting social and psychological difficulties of these patients must not be underestimated. The research workers are busy, and perhaps the patients can look forward to the development of an enzyme preparation, which, by replacing the missing enzyme, will revolutionise the treatment of galactosaemia, and enable them to live almost normal lives. However, until that happens, they must be content to feed on honey-dew and suchlike, and to avoid all milk, even that of Paradise!

"For he on honey-dew hath fed
And drunk the milk of Paradise."
(Samuel Coleridge Taylor: Kubla Khan.)

Acknowledgement

I should like to thank Dr. Charles Harris for permission to publish this case. Two of the patients mentioned in this account (J.L. and S.D.) have been described by Dr. A. White Franklin in the *Proceedings of the Royal Society of Medicine*, September, 1957, Vol. 50, No. 9, pp. 2 and 3.

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The Augustine Society

On 30th October, Father Bernard of the Society of St. Francis talked to the Augustine Society on the subject of Church unity. The purpose of seeking unity, he said, should be primarily not to strengthen the Church but to end the division of the Body of Christ.

The state of the Church is shown symbolically in the Church of the Holy Sepulchre at Jerusalem where a number of different denominations worship entirely separately. Because of their squabbling, the keys of the church are held by a Moslem family and much-needed restoration work is held up.

Division of the Church is often political in origin, as is the split in the Russian Orthodox Church to-day, but the chief cause is sin, pride, worldliness and small mindedness on each side. Luther and Wesley were both, in fact, quite orthodox theologians.

Hopeful signs are, however, to be seen. Ideas and movements originating in one part of the Church are tending to spread widely into the other parts. The Methodist view of the position of the laity is spreading in such a way; the Protestant churches are founding religious communities, and in the Roman Catholic Church, there is a renewed understanding of the importance of the Bible and a desire, in the Liturgical Revival, to reveal more clearly the basic meaning of the Eucharist, the vernacular movement is strong in France.

How can we personally work for unity, then? By prayer, by meeting other Christians and by the exchange of thought with them. There is no place for bargaining.

Fr. Bernard commended to us the Week of Prayer for Christian Unity during which

the prayer is for unity " as Christ wills " (January 17th-24th).

One of the obstacles to unity is the tendency of each group to caricature the others in order to boost its own morale. We must " meet to know to love ".

Finally we must, by listening to the beliefs of others, build up a composite picture of Christ.

A lively discussion followed, touching on the position of bishops, intercommunion (which Fr. Bernard thought should be an expression of unity and not a means to it), women in the ministry and the importance of being a faithful member of one's own church.

The Augustine Society was delighted to welcome several members of other religious societies. We hope they will come again.

LETTER TO THE EDITOR

The " Bleep " System

Dear Sir,

The " bleep " system has been in operation in the Hospital for over a year now, and an appraisal of its efficacy is, I think, due.

For those who are unaware of its existence, the bleep is carried by House Officers and other hospital officials who may be required urgently. It is a metallic instrument which clips easily in the pocket and emits a sound which is euphemistically known as a " bleep ". When a person is thus summoned, a phone call to the exchange enables a message to be given.

In theory, the bleep system is the Hospital Administrator's dream. It is difficult to ignore its persistent call; it has a wide range (the Bart's bleeps reach the proximal end of the White Hart bar), and the onus of responsibility is definitely on the receiving end.

At Bart's however, this excellent system seems to have failed. At peak bleeping hours, owing to overloading of the busy operators at the Telephone Exchange, it may take anything up to 20 minutes before the strident summons goes forth. To a caller on the " National " phone (including the interval before the exchange even answers the call), the time lag may approximate to 25 minutes. This fantastic situation seldom occurred in the pre-bleep era, when a reasonably conscientious House Officer marked the board in Surgery where he was likely to be found; an appropriate call from Surgery resulted in a message being delivered in reasonable time. Even more trying is the situation which may result when the bleep's battery runs dry, or the unfortunate instrument is otherwise indisposed. As there seems no way of telling, from the exchange end, when an instrument is working or not, and having had no answer from the bleeped one, the caller is told that the Doctor is not in the Hospital as he is " not answering his bleep ". In many cases no further effort is made to trace the missing person. A further objection may be made on aesthetic grounds. The sound made by a freshly-charged bleep is very terrible indeed, and if an unsuspecting patient is within range the results may well be disastrous. Similarly, the croaking of an ageing bleep may be the last straw in a busy day.

Bart's has always been suspicious of change (what other London Hospital can boast the fact that not even internal phones are fitted in the rooms in R.S.Q. ?), and the failure of the bleep system justifies this suspicion; it has, I suggest, been a dismal failure.

A return to the " bad old days " is clearly called for.

Yours sincerely,

R. P. Bonner-Morgan.

SPORTS NEWS

Rugby Club

1st XV v. C.U.L.X. Club. Lost 24-3.
18th Oct., 1961.

The Hospital went up to Cambridge with a somewhat weakened team and on the cold windswept Corpus rugger ground the LX Club easily defeated their XV who were outplayed in almost every department except, perhaps, in the tight. The LX Club were heavier and faster and although by half time with the score at 14-3 hope of victory was rather left behind (as was one member of the team later that evening) Bart's played hard to the finish, and only two scores were added to the Cambridge Club's victory in the second half. Had the Bart's covering been better the defeat, perhaps, would have been lessened, but it is difficult to see how it could have been avoided.

Team: A. P. Ross, R. V. Jeffreys, A. T. Letchworth, P. A. R. Niven, S. G. Harris, E. D. Dorrell, D. Chesney, O. J. A. Gilmore, B. H. Gurry, A. J. S. Knox, D. J. Delany, B. R. H. Doran, M. C. Jennings (capt.), C. J. Smart, D. Goodall.

1st XV v. O. Blues. Lost 14-3.
21st Oct., 1961.

By dint of intelligent use of their strong pack and defensive backs, the O. Blues won this game by 1 goal 2 trys and a penalty goal to a try, although it looked at half time as if the Hospital might well win despite a 3-point deficit. The Bart's pack were somewhat outplayed, but were far from being overwhelmed, and of the two sets of backs theirs seemed definitely superior. The Hospital try came when Doran, picking up a loose ball, started a movement which Jeffreys finished. However, in the second half two tries were kindly offered to the O. Blues and they gratefully accepted them, winning the match by a greater margin than the general run of play might have indicated.

Team: P. A. R. Niven, R. V. Jeffreys, J. E. Stevens, A. T. Letchworth, S. G. Harris, E. D. Dorrell, D. Chesney, J. W. Hamilton, B. H. Gurry, A. J. S. Knox, B. R. H. Doran, D. J. Delany, R. P. Davies, M. C. Jennings, C. J. Smart.

1st XV v. United Industries. Won 27-0.
28th Oct., 1961.

On a fine autumn day at Chislehurst the Hospital won this game easily with tries from Harris (2), Niven (2), Letchworth, Jeffreys and Jennings, but it was not until the second half that Bart's discovered that by throwing the ball about and by determined running tries were not difficult to get. Two tries, which resulted from orthodox line movements, were the only scores in the first half, but in the second, home supporters in any case must have enjoyed some entertaining play, especially when Niven joined the line. This win was matched by the 34-0 defeat of Esher by the "A" XV.

Team: P. A. R. Niven, R. V. Jeffreys, J. E. Stevens, A. T. Letchworth, S. G. Harris, E. D. Dorrell, D. Chesney, J. W. Hamilton, B. H. Gurry, A. J. S. Knox, B. R. H. Doran, D. J. Delany, R. P. Davies, M. C. Jennings (capt.), C. J. Smart.

CORNISH TOUR

1st XV v. Penzance. Lost 15-5.
4th Nov., 1961.

A strong Penzance XV made little impression on the Bart's defence for the first three-quarters of the game, with Jeffreys quick to recover the ball from the long diagonal kick of which the Penzance fly-half made full use; and, in spite of opposition's dominance in the line out and loose, Gurry made sure the Bart's backs had their full share from the tight. In reply to a penalty, Bart's scored almost immediately from the ensuing kick off with Harris breaking through a gap created by Jeffreys. At this stage the forwards were playing with rewarding determination, but the greater experience and weight of the home pack told towards the end. Gilmore, Doran and Jennings all played well in this game.

Team: P. A. R. Niven, R. V. Jeffreys, J. E. Stevens, A. T. Letchworth, S. C. Harris, E. D. Dorrell, D. Chesney, O. J. A. Gilmore, B. H. Gurry, A. J. S. Knox, B. R. H. Doran, D. J. Delany, A. P. Ross, M. C. Jennings (capt.), C. J. Smart.

1st XV v. Falmouth. Lost 6-0.
6th Nov., 1961.

Falmouth have enjoyed quite a successful season in Cornwall and Devon, although they possess no outstanding players, and Bart's did nothing to diminish this success in rather a dismal game under flood-lights. The Hospital's attack lacked cohesion at forward, half and back and Falmouth, making use of the tights and rather scrappy play, scored two tries, one in each half.

Team: P. A. R. Niven, R. V. Jeffreys, J. E. Stevens, A. T. Letchworth, S. C. Harris, E. D.

Dorrell, D. Chesney, O. J. A. Gilmore, B. H. Gurry, A. J. S. Knox, B. R. H. Doran, M. M. Orr, C. M. Cripps, C. J. Smart, M. C. Jennings (capt.).

1st XV v. Dartmouth B.R.N.C. Won 11-3.

Although the opposition was not very strong, this was a very encouraging game, where after 20 minutes the Hospital forwards gained the mastery which they retained except for a few minutes near the end, for the rest of the game. Furthermore, this possession, with Orr dominating the lineout, was not wasted in the backs, where both Harris and Sidebottom ran with considerable determination. Niven at full-back, where he played safely in defence, showed some power running in attack and Smart was prominent in the loose. With this win the tour ended on a very pleasant note.

Team: P. A. R. Niven, R. V. Jeffreys, E. S. Sidebottom, A. T. Letchworth, S. G. Harris, E. D. Dorell, D. Chesney, R. J. Shearer, B. H. Gurry, A. J. S. Knox, D. J. Delany, M. M. Orr, C. M. Cripps, C. J. Smart, M. C. Jennings (capt.).

1st XV v. O. Haberdashers. Lost 14-6.
11th Nov., 1961.

After 36 hours of rain, the pitch at Chislehurst was in surprisingly good condition when O. Haberdashers beat a rather jaded Hospital XV by 14 pts. to 6. The Bart's points came from a penalty by Harris from 5 yds. inside his own half, and a good try by Gurry from a grab cross-kick at the end of the game. Halls, playing for the 1st XV for the first time this season, had a good game, and Smart also was prominent, but when, towards the end of the game Bart's began to regain form, it was too late.

Team: P. A. R. Niven, R. V. Jeffreys, J. E. Stevens, A. T. Letchworth, S. C. Harris, E. D. Dorrell, D. Chesney, N. Greenwood, B. H. Gurry, A. J. S. Knox, B. R. H. Doran, M. M. Orr, M. C. Jennings (capt.), C. J. Smart, G. J. Halls.

1st XV v. O. Alleynians. Drawn 3-3.
18th Nov., 1961.

This was a fast open game which Bart's were perhaps a little unlucky not to win. In the first half in which the only score was a penalty goal to the O. Alleynians, Bart's were pinned to their own half by some superb touch kicking by the opposition's full-back. But in the second half Delany and Orr were working well in the tight and lineouts, Letchworth playing for the first time at fly-half, was carving great gaps in the Alleynian defence and Harris had three very fine runs, one of which resulted in the Hospital try. The back row of Jennings, Smart and Halls looked as cohesive as it has done this season. In spite of some violent assaults on each other's lines, neither side scored again before the final whistle.

Team: P. A. R. Niven, R. V. Jeffreys, J. E. Stevens, E. Sidebottom, S. G. Harris, A. T. Letchworth, D. Chesney, J. W. Hamilton, B. H. Gurry, A. J. S. Knox, J. Delany, M. M. Orr, M. C. Jennings (capt.), C. J. Smart, G. J. Halls.

Soccer Club

St. Bart's 1st XI v. Trinity Hall.
9 Nov., 1961
Result: Bart's 2. Trinity Hall 5.

Bart's began well against a strong Trinity Hall eleven with two Cambridge University players, but the Hall deserved their goal late in the first half. M. Waterworth brought us back on level terms with a finely anticipated goal just before the interval. In the second half Trinity Hall, with their superior attack, put the Bart's defence under continual pressure and scored three more fine goals. Bart's rallied, however, and reduced their lead with one of the many scoring chances the defence and inside forwards had worked hard to produce.

Team: B. Perriss, G. Haig, A. Howes, J. Pemberton, P. Savage (capt.), M. Hudson, E. Manson, H. Phillips, P. Herbert, M. Waterworth, N. Davies.

St. Bart's 1st XI v. King's College
Result: Bart's 5. King's College 1.

On a wet and miserable afternoon Bart's produced an excellent brand of football. P. Stanley opened the scoring for Bart's after some hard-fought mid-field play. P. Herbert made it two-nil for Bart's with an excellent individual effort, driving his shot from well out, just inside the post. Soon after half-time King's replied with a move that was destined to provide a goal; the outside right scoring from close range. H. Phillips and a penalty by Herbert put Bart's in an invincible position with two more goals. A fifth goal by Herbert rounded off a most successful effort by the whole team. Both N. Offen and T. Guthrie played well as newcomers in the half-back line.

Team: B. Perriss, A. Howes, T. Guthrie, N. Offen, P. Savage (capt.), M. Hudson, E. Manson, H. Phillips, P. Herbert, M. Waterworth, P. Stanley.

St. Bart's 1st XI v. Old Chigwellians.
Saturday, 18th Nov., 1961
Result: Bart's 1. Old Chigwellians 1.

This was a good match throughout with neither side easing under the pressure. B. Perriss stopped some dangerous early raids by the Chigwellians and the defence settled down quickly, managing to break up a lively attack. N. Offen covered well at left half, and B. Hore and G. Haig worked well together as the last line of defence. Changing over with no score, Bart's then showed more determination than usual. Eventually P. Herbert scored a fine goal which the useful Chigwellian goalkeeper had no chance of saving. Although the defence held on grimly they were unable to stop one of the late Chigwellian moves culminating in a well-deserved goal.

Team: B. Perriss, A. Howes, G. Haig, J. Pemberton, B. Hore, M. Hudson, P. Stanley, P. Herbert, N. Offen, H. Phillips, N. Davies.

Hockey Club

Wed. 25th Oct.—Fri. 27th Oct., 1961

The Hockey Club's visit to Cambridge this season was reverted to its accustomed autumnal position in the fixture list, and the weather more than repaid its debt for February's washout !

On the Wednesday we played Pembroke College under ideal conditions. The first half produced some fast play with Bart's taking the territorial advantage. Constructive moves among the forwards invariably ended scrappily in the circle where finishing power was not apparent. Our strength in short corners soon registered a score, but shortly after half-time S. Phillips, in goal, bravely stopped a rising shot, only to fall with a fractured right radius. This incident, together with revived Pembroke pressure, told on the beleaguered Bart's defence. The final score was 2-4.

On Thursday, we played King's College. The last-minute arrival of the Captain and the second-string goalkeeper restored morale and in the opening minutes it was clear we were to have most of the game our way. Constructive passing among the halves and forwards kept us near the opposition's goal, but lack of aggressive play and thrust in the circle left most of the movements unrewarded. It seemed that Bart's were quite satisfied with their three-goal lead and towards the end play became slow and scrappy.

In contrast the Friday match against Jesus College, whom we had beaten the previous Saturday, was a battle to the final whistle. With their side strengthened, Jesus set the fast pace, although the ground had had a soaking during the night.

Plentiful Bart's forward movements were frustrated by a tight opposing defence. In the second half, when the score was level 2-2, the Bart's defence was caught and a swerving shot, baffling the goalie, just made the net. Indignant at conceding the lead, Bart's pressed even harder for the closing quarter hour, unfortunately without success.

Those who played were: S. Phillips, S. Campbell-Smith, W. H. Pagan, A. Frank, C. D. R. Flower, A. R. Robertson, R. Courtenay-Evans, S. Thomas, M. Smith Walker, A. Edelston, P. J. Kingsley, D. Glover, P. W. Caine, W. Castleden.

Swimming Club

The United Hospitals Water Polo League is being played this term at St. Mary's and Bart's have entered two teams.

Barts I v. St. Thomas' I. Won 10-5.

The side built up last year started this new season with a convincing win over St. Thomas'. Shorey had little trouble in beating his man and accounted for five of the Bart's goals, and was well supported by Ruoss, Groves and Shand. The goalkeeper remains the only permanent unfilled position in our team and despite a good defence the opposition managed to net the ball five times.

Barts I v. St. Mary's II. Won 6-4.

The team, without Groves for this match, due to illness, made very heavy weather of beating a Mary's side who failed to conceal their fouling from the referee, resulting in three successful penalty shots to Bart's. The rather rough game tends to upset the side and more advantage should have been taken of the fairly permanent absence of one of the opposing team.

Bart's II v. Middlesex II. Won 12-2.

This was a scrappy match doing neither side great credit, out of which Bart's emerged victorious because of two members of the side who could shoot at goal hard and accurately.

Bart's I v. London I. Won 10-4.

There was a very noticeable improvement in the side after some intensive training in the nurses' pool, resulting in much greater cohesion and the opposition were outmanoeuvred for most of the match. Their goals came only from a tendency to mark closely on our part when winning and if this was overcome we should have little difficulty in reducing the number of goals scored against us.

Cross Country

So far this season Bart's have run in two matches in the first division of the University Cross-Country League. Competition is stiffer than it was last year when we won the 2nd Division; at the moment we are lying 2nd to Imperial College who are extremely strong, having 15 runners in the University teams. Although many consider this sport to be a highly individual one the results of these two matches and of those to come will depend largely upon the weaker members of the team rather than the first two men home.

Placings in 1st Match		Placings in 2nd Match	
2nd	Littlewood	1st	Littlewood
4th	Foxton	4th	Foxton
20th	Pott	6th	Pott
67th	Phipps	31st	Lewis
78th	Lewis	33rd	Saunders
84th	Hardy	39th	Hardy
96th	Pickard	53rd	Pickard
137	Finished	61	Finished
4th posn. in League		2nd posn. in League	

Apart from these matches all members have been running in U.H. matches and are getting fitter. Littlewood won the University trials and has represented the 1st eight in all its matches so far, he has also run for U.H. against Mitocavians and Orion, and again against Sandhurst and University of London "A", breaking two course records. Foxton has been hard on his heels and also ran for the University v. Oxford and v. Cambridge, coming 10th on both occasions. He further distinguished himself by recording the fastest lap in the U.C. relay when a weathered Bart's team came 24th. Pott, in spite of everything, is getting fitter and running well in heavy conditions which hold better things back!

P.L.

BOOK REVIEWS

Histology by Arthur W. Ham and Thomas Sydney Leeson. 4th Edition. Pitman Medical Publishing Co. 85s.

This book has established an excellent reputation, which the latest edition fully supports. There has been extensive alteration to the chapters dealing with the cell and also in the description of special methods. It is a pity that the erroneous diagrams of the paths of rays through the light and electron microscopes still retain their errors. The only other blemishes are that the fluorescent antibody technique of Coons and Caplan uses fresh frozen sections and that these are not fixed until after the fluorescent antibody has been applied and, indeed, may never be fixed in the ordinary sense of the term. These, however, are minor quibbles; the book can be wholeheartedly recommended to anyone who can firstly pay the price, and secondly face the rather forbidding bulk.

The style is clear and very readable so that the size is somewhat less of an obstacle than would be expected. It is still sufficiently great to deter most undergraduates, but as a reference work for the benefit of research workers and teaching staff it is most valuable. F.J.A.

Roxburgh's Common Skin Diseases by P. F. Borrie. Published by H. K. Lewis and Co., London. £1 17s. 6d.

To the harassed medical student and practitioner this textbook will be most welcome in giving him an elementary grasp of dermatology. This almost entire revision is generally well-illustrated, clearly printed on good-contrast paper, and the subject matter concisely dealt with—above all, it is easy to read and to understand.

The early chapters of basic morphology are recommended and, in addition, there is a good chapter on general treatment in which, for instance, the indications for corticosteroid therapy are carefully outlined, on a rational basis, and provide a good working rule in making the decision to use these drugs. Perhaps one of the most difficult conditions for the student to understand is the eczema-dermatitis group. A lucid expression of the diagnostic group is given here, particularly in tracing the development of the lesions in its various phases. There is also a working summary of the various factors both specific and non-specific, which affect the skin prone to eczema. It is rarely appreciated that the skin which has been subjected to repeated trauma from various irritants seldom escapes without permanent damage, and requires continued care even when the acute phase has settled.

In discussing dermatitis medicamentosa, an unfortunate error may confuse the reader. Correctly speaking, dermatitis venenata is the term which should be used to describe allergic eruptions due to the topical application of medicaments, while the diagnosis of dermatitis medicamentosa synonymous with drug eruption should be reserved to indicate those eruptions due to the internal administration of medicaments. A helpful list of various drugs and the characteristic eruptions which they may induce appears on pages 382 to 384.

Throughout the remainder of the text much emphasis is placed upon the association between skin eruptions and systemic disorders—a feature whose importance is more and more realized by all imaginative physicians.

Erythema nodosum, which used to be regarded as almost invariably tuberculous in aetiology, is now seen to appear with streptococcal infections, sarcoidosis, ulcerative colitis, drug eruptions, and only rarely from acid-fast infection.

Simple therapeutic measures have been appended to each disease discussed which makes the separate topics complete in themselves and readily available for rapid reference even in the "surgery". Finally, that big "bug-bear" of differential diagnosis is emphasised with each discussion. A handy book for study. A.S.

Ophthalmic Operations by Seymour Philps. Ballière, Tindall and Cox. Price 70s. Second Edition by John Foster.

The author of this handsomely-produced book has especially in mind, we are told in the Preface, the F.R.C.S. candidate and the ophthalmic surgeon who operates only occasionally. The latter will no doubt be happy with the book, and safe in its hands, but the F.R.C.S. candidate will need to supplement it extensively. Indeed, he is warned in the Preface that the book is, at the time of publication, in some fields behind the times. Unfortunately, this warning has not been carried far enough, and the F.R.C.S. candidate who retains the impression that a satisfactory method of lens extraction for ordinary use is one employing a complete iridectomy and no corneo-scleral sutures will start with a handicap. (He should, furthermore, avoid the capsule grip at six o'clock in the "Kirby" method!) He would, again, be most unwise to exile the prophylactic peripheral iridectomy in angle-closure glaucoma to the United States of America.

The surgical treatment of retinal detachment has been confined to the diathermy operation, scleral shortening and vitreous implantation. The space devoted to the treatment of uniocular aphakia by acrylic implants—a method having few remaining adherents—might with advantage have been given over to those newer techniques in retinal detachment work which are being used more and more extensively. Scleral implantation, encircling tubes and sutures, photo-coagulation and the Custodis method of treatment should be described in a book of the scope intended. J.E.C.

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